

# The Ontario Air Pollution Study: Identification of the Causative Agent

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Previously published data from the Ontario Air Pollution study are reviewed. It has been shown that there is a consistent association in summer between hospital admissions for respiratory disease in Southern Ontario, and daily levels of  $\text{SO}_4$ ,  $\text{O}_3$ , and temperature. No association exists for a group nonrespiratory conditions. Multiple regression analyses are presented that show all environmental variables account for 5.6% of the variability in respiratory admissions and that if temperature is forced into the analysis first, it accounts for 0.89% of the variability only. Distribution plots of standardized residuals are presented. In June of 1983, there were an exceptional number of ozone episodes (defined as occasions when ozone was  $> 82$  ppb for 3 or more hours in a calendar day) in this region. A separate analysis of hospital admissions for acute respiratory diseases for the month of June for several years shows no demonstrable excess in June of 1983; previously regional analyses have indicated that ozone is associated with increased levels in July and August over a 9-year period. It has also been found that daily  $\text{SO}_4$  data collected at one monitoring site in the center of the region are not correlated with respiratory admissions, whereas the  $\text{SO}_4$  values collected every sixth day, on different days of the week, at 17 stations in the region had the highest correlation with respiratory admissions. It is concluded that probably neither ozone nor  $\text{SO}_4$  alone is responsible for the observed association with acute respiratory admissions, but that either some unmeasured species (of which  $\text{H}_2\text{SO}_4$  is the strongest candidate), or some pattern of sequential or cumulative exposure is responsible for the observed morbidity.

## Introduction

This study began in 1978, and the first publication was in 1983 (1); the data and analysis were extended in a communication to the second U.S./Dutch International Symposium in May 1985, which was published in 1986 (2). The data was brought up to date in a recent paper in *Environmental Research* published in August 1987 (3). The purpose of this presentation is to discuss additional data and analyses that bear on the important question of which air pollutant is most likely to be responsible for the demonstrated respiratory morbidity.

## Summary of Previous Data

The three papers already published have shown that there is a consistent association over the period between 1974 and 1983 between temperature, ozone ( $\text{O}_3$ ), sulfate ( $\text{SO}_4$ ), and, to a lesser extent, sulfur dioxide ( $\text{SO}_2$ ), in the summer only, and excess hospital admissions for

acute respiratory conditions in Southern Ontario. Previously published data also show that there is no relationship of a group of nonrespiratory conditions to any pollutant. Each day is compared only to the average admissions for that day of the week in that season in 1 year, thus avoiding the confounding produced by long-term changes in admissions. Dividing the region into nine subregions and associating each sampling station with a group of adjacent hospitals, it is possible to show that respiratory admissions on high ozone days (80–199 ppb) are greater by about 7% than admissions on low ozone days (ozone levels 9–60 ppb), comparing only the same days of the week in the same season in the same year (3). Although asthma admissions constitute nearly half of all summer respiratory admissions (3), the correlation coefficients remain highly significant if asthma is taken out of consideration.

The Ontario Air Pollution study includes all the 79 hospitals in the region that admit acute cases and the hourly pollutant data for  $\text{SO}_2$ ,  $\text{O}_3$ ,  $\text{NO}_2$  (nitrogen dioxide), CO (carbon monoxide), coefficient of haze, and  $\text{SO}_4$  measurements conducted over a 24-hr period every sixth day at each of 17 stations are used for the estimate of pollutant level.

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## Multiple Regression Analyses

Equation 1 represents the variables that show a significant Pearson correlation coefficient to respiratory admissions in the summer. These account for 5.6% of the variance in respiratory admissions.

$$\text{PDYTRP} = -7.10 + 1.146*(\text{T.L48}) - 0.552*(\text{T.LO}) - 0.766*(\text{T.L24}) + 0.015*(\text{SO}_4\text{.L48}) + 0.017*(\text{SO}_4\text{.LO}) + 0.049*(\text{SO}_4\text{.L24}) + 0.044*(\text{O}_3\text{.LO}) - 0.071*(\text{O}_3\text{.L48}) - 0.009*(\text{O}_3\text{.L24}) \quad [1]$$

$$R^2 = 0.056 \text{ (5.6\% of variance)}$$

where PDYTRP = percentage deviation in respiratory admissions adjusted by the mean for the same day of the week in the same year; LO = same day; L24 = lagged 24 hr; L48 = lagged 48 hr.

As noted in previous publications (3), there are high correlation coefficients between these variables. Between  $\text{O}_3$  and  $\text{SO}_4$ , 0.6459; between  $\text{O}_3$  and temperature, 0.6450; and between temperature and  $\text{SO}_4$ , 0.5237. Relative humidity is correlated only with  $\text{SO}_4$ , with a correlation coefficient of 0.3445. All of these coefficients were significant at the  $p < 0.001$  level (3).

In a further multiple regression analysis, temperature was forced into the analysis as the first variable. As shown in Table 1, it accounted for only 0.89% of the variation. When  $\text{SO}_4$  was inserted as step 2, 3.3% of the variability was accounted for, and this increased to 5.6% when all other variables were added. This suggests that temperature alone is not the responsible factor for the association.

Figure 1 shows the distribution plot of the standardized residuals for the summer data only. These show a satisfactory fit between the observed and predicted distribution.

## Data for June 1983

The usual data analysis in this study involved only the summer months of July and August. However, June 1983 was observed to be a month with particularly high ozone levels. As shown in Table 2, ozone episodes in this region, defined as ozone levels above 82 ppb for 3 or more hours in a calendar day, were more frequent in June 1983 than in any July or August. This table shows the variability between years of these episodes. In view of the data for June 1983 we obtained the hospital admission data for the month of June only for the years between 1979 and 1985. These are shown in Table 3. Surprisingly, in view of the regional analyses of high and low ozone days we have previously reported (3), there is no excess of respiratory admissions in June 1983. There were more in June of 1979, 1980, 1982, and 1985 than there were in June 1983. As noted later, this observation throws doubt on the primacy of ozone in relation to the morbidity.

Table 3 is also interesting as it shows that in years when hospital admissions in June were highest (1982 and 1985), the increased admissions were in the categories of acute bronchitis and asthma, and not in the other respiratory categories.

Table 1. Ontario air pollution study multiple regression analysis, July 1987.

Method	$R^2$
Forcing T.L48 into analysis as first variable	0.0089
Inserting $\text{SO}_4\text{.L24}$ as step number two	0.03329
Adding all other variables	0.056

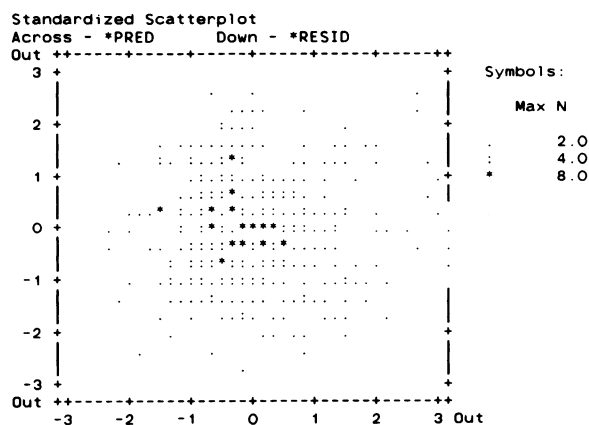
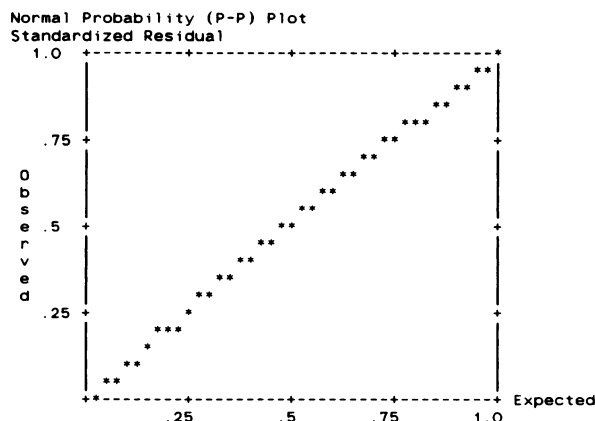


FIGURE 1. Standardized residuals from multiple regression analysis, summer data only.

Table 2. Station ozone episodes by year and month.<sup>a</sup>

Year	April	May	June	July	August	September
1976	6	10	76	24	71	33
1977	13	68	37	92	20	4
1978	—	95	72	117	124	23
1979	2	14	64	99	5	15
1980	1	20	58	49	26	7
1981	—	37	39	74	36	10
1982	3	8	8	33	18	7
1983	—	—	140	52	35	28
1984	—	2	32	63	44	9
All years	25	254	526	603	379	136

<sup>a</sup>Station episode =  $\text{O}_3$  above 82 ppb for 3 or more consecutive hours in a calendar day. Data from Davis et al. (5).

## Use of Daily SO<sub>4</sub> Data Collected at One Station

In our study, we used the SO<sub>4</sub> data for the region recorded at 17 stations for 24 hr every sixth day. We learned that a separate monitoring station had been established in 1979 at Long Point, near London, Ontario, as part of the Acid Rain Research Programme of Environment Canada. Tapes of these data were made available to us. In summer, observations of SO<sub>4</sub> were correlated with the SO<sub>4</sub> data we have used for the whole region, with a coefficient of 0.6057, highly significant at the 0.001 level. We then reran the correlative data with hospital admissions and noted that whereas the SO<sub>4</sub> data from the whole region were highly significantly correlated with the respiratory admissions (3), the daily SO<sub>4</sub> data from the single station were not correlated significantly with the admissions. We conclude from this that the regional spread of the SO<sub>4</sub>, as represented by the 17 stations, is important and cannot be replaced with a single data point from the center of the region.

## Summary of Evidence for and against Different Pollutants

In Table 4, we have summarized the points that can be made for and against individual pollutants. It is clear that a case can be made that O<sub>3</sub> is responsible; not only is it the only one of the measured pollutants that

has been shown to be irritant at the ambient concentrations which exist (4), but also the regionally divided data showed a difference between hospital admissions on high and low O<sub>3</sub> days. Yet the analysis of data for June 1983, when ozone levels were unusually high, showed no evident increase in hospital admissions. The multiple regression showed that SO<sub>4</sub>, which had the highest Pearson correlation coefficient with respiratory admissions, accounted for the highest percentage of the variability. These factors favor attributing the effect to the SO<sub>4</sub> levels, yet sulfates are not irritant at these concentrations. Nor can any association between SO<sub>4</sub> levels and respiratory admissions in winter be demonstrated.

These conflicting results suggest that some other pollutant, the formation of which is favored by high O<sub>3</sub> and SO<sub>4</sub> levels, might be responsible. A possible candidate is H<sub>2</sub>SO<sub>4</sub> aerosol, which, as noted in our most recent publication (3) has been measured in this region. More aerometric data will be required to confirm or disprove this possibility. An initial analysis of hospital admissions on the days in July 1986 when H<sub>2</sub>SO<sub>4</sub> was measured will be undertaken when the hospital data are available.

We conclude that this question cannot be resolved on the basis of present data. It may be that the morbidity is produced by some combination of factors. Ozone may increase the airway reactivity; if the subject is then exposed to acidic aerosol, or even naturally occurring antigens, the response is heightened sufficiently to lead to hospital admission. It may prove to be important that in this region the pollutants are encountered by a

Table 3. Respiratory admissions to 79 hospitals, month of June only.

Diagnosis	1979	1980	1981	1982	1983	1984	1985	Total
Acute bronchitis	182	221	140	198	152	159	181	1,233
Viral pneumonia	25	19	12	11	13	8	9	97
Bacterial pneumonia	408	335	257	277	281	227	231	2016
Bronchitis	82	144	122	103	104	93	73	721
Chronic bronchitis and emphysema	210	178	167	160	154	144	135	1,148
Asthma	837	1,000	834	1,240	913	868	1,173	6,865
Totals	1,744	1,897	1,532	1,989	1,617	1,499	1,802	12,080

Table 4. Comparative evidence.

Factor	Points in favor	Points against
Temperature	Significant Pearson correlation coefficient; effect seen only in the summer	On regression analysis, accounts for only 0.89% of the variance in respiratory admissions
Ozone	Related to asthma attacks; present in high concentration only in summer; regional data show effect of high ozone days on admissions; known to be irritant at these concentrations	SO <sub>4</sub> has higher Pearson correlation coefficient; no increased hospital admissions in June 1983 when O <sub>3</sub> levels were high; asthma is not the only diagnostic category involved
Sulfates	Highest correlation with respiratory admissions; asthmatics are known to be sensitive to SO <sub>2</sub> and SO <sub>4</sub>	Not known to be irritant at these concentrations; no winter relationship to respiratory admissions
H <sub>2</sub> SO <sub>4</sub> or other species not routinely measured	Formation of aerosol H <sub>2</sub> SO <sub>4</sub> has been demonstrated in this region; formation likely when both O <sub>3</sub> and SO <sub>4</sub> are high	Frequency of formation not yet known; not shown to be irritant at ambient levels so far measured

population unacclimatized to them, with a consequently increased response. More data will be required before this question can be resolved; a detailed analysis of the actual cases admitted to hospital on a typically high  $O_3$  and  $SO_4$  day might shed more light on the association we have demonstrated.

## Summary and Conclusion

We draw the following conclusions from the data as it now stands.

The association between the difference between respiratory admissions on a given day and the mean for that day of the week, for that season, in that year, and  $O_3$ ,  $SO_4$ , and temperature, can be consistently demonstrated in the summer. No association exists for a group of nonrespiratory conditions.

Multiple regression analysis indicates that temperature alone accounts for less than 1% of the variance;  $SO_4$  accounts for about 3%; and when those factors together with  $O_3$  are included in the analysis, 5.6% of the variability in respiratory admissions is accounted for. However, there are reasons against attributing the association either to  $O_3$  or to  $SO_4$ . It is possible that the morbidity is attributable to a pollutant such as  $H_2SO_4$  aerosol, which has been shown to be formed intermittently in the summer in this region. As this is not yet routinely measured, the present type of analysis cannot be conducted in relation to it.

From what is known of individual susceptibility to these pollutants from clinical studies, it is possible that

ozone heightens airway reactivity, rendering the individual susceptible to subsequent  $SO_4$  or  $H_2SO_4$  exposure; the possibility that these pollutants heighten sensitivity to other (unmeasured) allergens cannot be ruled out. A high correlation coefficient between such allergens and  $O_3$  and  $SO_4$  data would be required to attribute the association with morbidity to such allergens.

Measurement of  $SO_4$  at a single station in the center of the region, though highly correlated with the measurements we have used from 17 stations, cannot be used in place of the regional  $SO_4$  data, as it shows no significant association with hospital admissions.

## REFERENCES

1. Bates, D. V., and Sizto, R. Relationships between air pollutant levels and hospital admissions in Southern Ontario. *Can. J. Public Health* 74: 117-122 (1983).
2. Bates, D. V., and Sizto, R. A study of hospital admissions and air pollutants in Southern Ontario. In: *Aerosols: Research, Risk Assessment and Control Strategies* (S. D. Lee, T. Schneider, L. D. Grant, and P. J. Verkerk, Eds.), Lewis Publishers, Inc., Chelsea, MI, 1986, pp. 767-777.
3. Bates, D. V., and Sizto, R. Air pollution and hospital admissions in Southern Ontario: The acid summer haze effect. *Environ. Res.* 43: 317-331 (1987).
4. McDonnell, W. F., Chapman, R. S., Leigh, M. W., Strobe, G. L., and Collier, A. M. Respiratory responses of vigorously exercising children to 0.12 ppm ozone exposure. *Am. Rev. Resp. Dis.* 132: 875-879 (1985).
5. Davis, C. S., Reid, N. W., Heidorn, K. C., and Ormrod, O. P. Final Report: Oxidants Assessment: Ontario Data Base Phase 1. Environment Canada, Ottawa, 1986.